

# Traumatic "Brain Stem Contusion" (BSC): Acute Presentation and Management in a Tertiary Medical Centre- A Case Report and Review of Literature

Pankaj Kumar Swarnakar<sup>1</sup>  
 Soubhagya Ranjan Tripathy<sup>2\*</sup>  
 Manmath Kumar Dhir<sup>3</sup>  
 Sanjib Mishra<sup>4</sup>  
 Sitansu Kumar Rout<sup>1</sup>  
 Bikash Ranjan Behera<sup>1</sup>

<sup>1</sup>Department of Neurosurgery, SCB Medical College, Cuttack, Odisha, India  
<sup>2</sup>Consultant Neurosurgeon, Shree Hospital, Lewis Road, Bhubaneswar, Odisha, India  
<sup>3</sup>Department of Neurosurgery, SCB Medical College, Cuttack, Odisha, India  
<sup>4</sup>Department of Neurosurgery, SCB Medical College, Cuttack, Odisha, India

## Abstract

### Introduction:

Traumatic brainstem lesion (BSC) after head injury due to RTA (Road Traffic Accident), though an uncommon event, depends upon the high velocity acceleration-deceleration forces. It leads to rise in Intracranial Pressure (ICP) & brain herniations, resulting in change in brainstem microstructure. Focal traumatic BSL is a rather rare event.

### Case Description

Here we present an interesting case of 35 year male presented to our E.D. (**Emergency department**) with two episodes of vomiting & altered sensorium, due to fall, with Glasgow Coma Scale (GCS) of E1V1M5 (7/15). There was left sided hemiparesis. Computed Tomography (CT) scan showed brain stem contusion. He was kept in Intensive Care Unit (I.C.U) for the monitoring of pulse, Blood Pressure (BP), temperature. After 17 days of conservative treatment, the patient recovered. Pt was followed up on monthly basis for next 3 months. The BSL resolved as was depicted on the repeat CT scans.

### Conclusion

The severe morbidity and mortality associated with BSC mandates management in ICU for round the clock monitoring. This adds to the crunch of bed occupancy in any Government sector tertiary centre, but nevertheless, the outcome of such monitoring is matchless in terms of improved survival & better Quality Of Life (QOL). These cases needs further study to evaluate their future outcome. This may be the first of its kind of case report (after extensive search in Pubmed, using different relevant keywords, amongst the English literature) on this prickly issue, while choosing between catering to all & providing best available care to all.

### Keywords

Brain Stem Contusion(BSC); RTA (Road traffic accident); Brainstem; Intensive Care Unit (I.C.U); Quality of Life (QOL)

### Introduction

Traumatic Brain Injury (TBI) constitutes a wide spectrum of clinic-radiological events, ranging from EDH (Extra dural hematoma), ASDH (Acute Subdural Hematoma), contusion or DAI (Diffuse Axonal Injury). TBI present to Emergency Department (ED) either due to RTA (Road Traffic Accident), an assault or a fall from height. They result in closed type or open type of head injury. Brain stem is the part of brain more over injured by severe collisional impact to the skull causing alteration in microstructural organization inside the brain. Patients usually present with altered sensorium. The impact of trauma may be either localized or distributed all over. Here we present an uncommon case of **traumatic "Brain Stem Contusion" (BSC)**, its presentation & management in an acute setting.

### Case Description

Here we present a case of a 35 year male (Figure 1b) presenting to our E.D. (**Emergency department**) with altered sensorium, vomiting, bleeding from nose and two episode of convulsions, due to fall from bike under the influence of alcohol, with Glasgow coma scale (GCS)- E1V1M5 (7/15). There was left sided hemiparesis (MRC- Medical research council, Grade-2/5).

Pulse rate was 62 per min, blood pressure 140/80 mm of Hg. Respiration was abdomino-thoracic type, 18 per minute; left pupil dilated but sluggishly reacting to light, right side normally reacting to light. There was right side weakness (Figure 1a) of both the limbs. Noncontrast CT scan of the brain revealed brain stem contusion (BSC), in the midbrain area on the right side with DAI (Figure 2).

## Article Information

**DOI:** 10.31021/jnn.20181105  
**Article Type:** Review Article  
**Journal Type:** Open Access  
**Volume:** 1 **Issue:** 1  
**Manuscript ID:** JNN-1-105  
**Publisher:** Boffin Access Limited  
  
**Received Date:** November 01, 2017  
**Accepted Date:** November 20, 2017  
**Published Date:** January 12, 2018

### \*Corresponding author:

**Soubhagya Ranjan Tripathy**  
 Consultant Neurosurgeon  
 Shree Hospital  
 Lewis Road  
 Bhubaneswar  
 Odisha, India  
 Tel.no:9861008487  
 E-mail:soubhagya.tripathy@gmail.com

**Citation:** Tripathy SR, Dhir MK, Swarnkar PK, Mishra S, Rout SK et al. Traumatic "brain stem contusion" (BSC): Acute presentation and management in a tertiary medical centre- A case report and review of literature. J Neurosci Neurosurg. 2018 Jan; 1(1):105

**Copyright:** © 2018 Tripathy SR, et.al. This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 international License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.



**Figure 1a:** RTA victim with right side hemiparesis



**Figure 1b:** Same patient

All routine blood investigations, liver function test, coagulation profile, renal function test, cardiological evaluation, Chest X Ray (CXR) and Ultrasonogram (USG) of abdomen and pelvis were within normal limit. The plantar reflex was up going, suggesting an UMN (Upper Motor Neuron) type of involvement.

The BSC started resolving & in the second NCCT brain, it has vanished; but DAI was persisting (Figure 3). The Marshall CT grade was 2. The patient underwent tracheostomy on post trauma day-3; for the need of proper ventilation (reduction of the anatomical dead space). The respiration remained regular and abdomino-thoracic type, since the day of admission.

The patient had unequal pupils; being dilated on left size, with sluggish reaction to light. The pupil size was measured with a pupilometer, by the ophthalmologist of our hospital. Patient was monitored regularly on daily basis: for, temperature, BP, PR, Respiratory rate and pupil size followed by the neurological assessment of motor system (Chart-1).

On post trauma day 4, we started giving adequate nutrition to the patient through the nasogastric tube. Relatives were counseled and taught about the conservative patient care. After day 20, the patient was discharged with medications. The neurological status improved, the pupils became equal with normal reaction to light; but isolated left side hemiparesis persisted. The patient is on follow up after that, on monthly basis for last 3 months. Barring left hemiparesis of MRC grade-2/5, he has fully recovered neurologically. CT scan of brain had no evidence of BSC on day-20 (Figure 4).

## Discussion

The most frequent site of haemorrhage is the midline rostral brainstem. When associated with Space Occupying Lesion (SOL) and increase in ICP, it's called Duret haemorrhage. In case of Diffuse Axonal Injury (DAI), dorso-lateral quadrant is the site of lesion. Diffuse vascular injury results in BSL in the periventricular areas.

BSC portend an ominous outcome with a poor prognosis. The severity of outcome depends on the contusion size (hematoma volume  $\geq 6$  ml); its site- midbrain, pons, medulla, or in toto; intraventricular extension; hydrocephalus. No definitive management protocol exists till now. Associated pathology may be dealt with, depending on the hematoma size; with presence or absence of mass effect. In our report, the patient had midbrain bleed, not involving the ventricle, and there was no associated hydrocephalus.

Hemiparesis is due to involvement of the corticospinal tracts and the left sided pupillary dilation can be explained as local pathology involving the long ciliary nerve [1].

Commichau et al., (2003) described that rise in temperature of body manifesting from a central cause leads to rise in morbidity and mortality rate, among patients with TBI. Greer et al., (2008) showed that, as an independent variable fever is consistently associated with worse outcome in patients with TBI. Deogaonkar (2005) illustrated that, the severity of fever caused by massive volume intracerebral hematoma is attributed to direct compression of brainstem and hypothalamic thermoregulatory centers. Wijidicks and St Louis (1997), in a retrospective study correlated mortality rate in patients associated with low GCS<8, absent brainstem reflex like oculocephalic reflex or poor motor response in brainstem hemorrhage [2].

Tintore M et al., concluded that infratentorial lesions increases the disability rate [3]. Today, though multimodal investigation facility is available still CT remains the reliable immediate imaging modality of choice for any TBI, the diagnostic features being contusion, obliteration of basal cistern, blood in prepontine or perimesencephalic cistern [4,5,6].

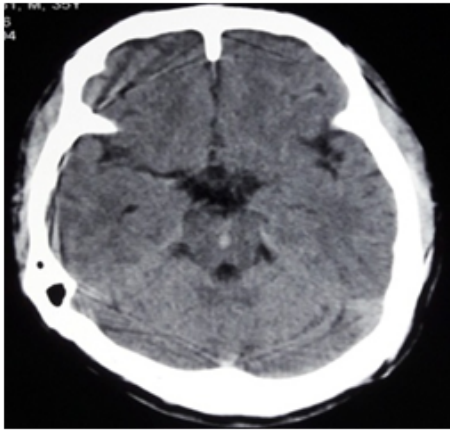
Biomechanics of BSC are: damage by direct impact, flexion and distortion and vascular involvement or hyperextension. Primary brainstem lesion (contusion or laceration) is due to movement of the brain along the sharp tentorial edges [7]. According to Cooper et al, acute flexion is the main recognized factor for BSC, contrary to hyperextension [8].

Usually, BSC patients present with low GCS, making ICU care with ventilator support, the need of the hour. Prompt care by neurosurgeons, nursing care and last, but not the least patient's own attendants' care are the pillars upon which this arduous task rests on. Attitude towards the "conservative" patient, matters the most. In resource poor public hospitals, these are the group of patients prone to the most neglect. Systemic changes like pulmonary complications,

Day	PR (/min)	BP (mmHg)	Temp	RR	Pupil Size (mm)	GCS	Reaction To Light
1	58	140/80	afebrile	14/min	Lt- 5 mm Rt- 3mm	E 3VtM 3	Lt- sluggish reacting RT-normal reacting
2	56	138/82	afebrile	14/min	Lt - 5mm Rt -3mm	E 3VtM 3	Lt - sluggish reacting RT-normal reacting
3	60	138/80	afebrile	16/min	Lt -5mm Rt -3mm	E 3VtM 3	Lt- sluggish reacting Rt-normal reacting
4	60	130/80	afebrile	14/min	Lt -5 mm Rt -3 mm	E 3VtM 3	Lt- sluggish reacting Rt-normal reacting
5	58	130/80	afebrile	14/min	Lt -5mm Rt -3mm	E 3 VtM3	Lt- sluggish reacting Rt-normal reacting
6	58	130/80	afebrile	14/min	Lt- 5mm Rt -3 mm	E 3 VtM3	Lt- sluggish reacting Rt-normal reacting
7	60	132/82	afebrile	16/min	Lt- 5mm Rt -3 mm	E 3 VtM3	Lt- sluggish reacting Rt-normal reacting
8	56	134/80	afebrile	16/min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
9	58	132/80	afebrile	14/min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
10	56	130/80	afebrile	16/min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
11	56	128/80	102 <sup>0</sup> F (febrile)	12/ min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
12	64	128/82	102 <sup>0</sup> F (febrile)	12/ min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
13	64	126/80	afebrile	12/min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
14	62	128/82	afebrile	14/min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
15	64	128/82	afebrile	14/ min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
16	68	124/80	afebrile	16/ min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting
17	68	124/80	afebrile	16/ min	Lt- 5mm Rt -3mm	E 3 VtM5	Lt- sluggish reacting Rt-normal reacting

**Chart-1:** The Daily Assesment Table**CT-** Computerised Tomography Scans**EDH-** Extradural Hematoma**DC-** Decompressive Craniotomy**ICP-** Intracranial Pressure**GCS-** Glassgow Coma Score**BP-** Blood pressure**QOL-** Quality of life**DAI-** Diffuse Axonal Injury**MRC-** Medical Research Council**USG-** Ultrasonography**NCCT-** Non Contrast CT Scan**SOL-** Space Occupying Lesion**Chart-2: Abbreviations****n-** Total number of cases**SDH-** Subdural Hematoma**BSC-** Brain Stem Contusion**ED-** Emergency Department**RTA-** Road Traffic Accident**ICU-** Intensive Care Unit**TBI-** Traumatic Brain Injury**ASDH-** Acute Subdural Hemorrhage**CXR-** Chest X Ray**UMN-** Upper Motor Neuron**DAI-** Diffuse Axonal Injury

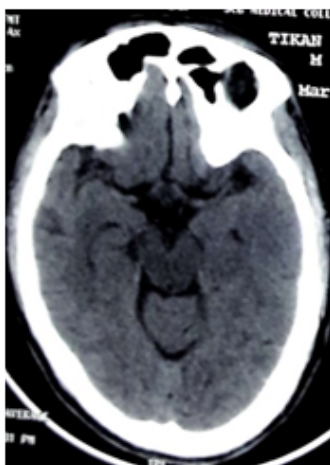




**Figure 2:** Brain Stem Contusion (BSC) in the midbrain



**Figure 3:** Resolving BSC with DAI on repeat scan with cisternal bleed



**Figure 4:** Fully resolved BSC on PTD-20

cardiological changes must be monitored, apart from the vital parameters. Associated injury to abdomen, thorax or any bony or soft tissue must involve general surgeon and orthopedicians. Of course spinal injury must be ruled out after deliberation on the brain lesion[9].

Mohapatra et al., reported survival in more than 50% of such cases. In their 70 patients study, 38 patients survived and 22 had a good recovery. Age, associated skull fracture and other associated injury continue to be the determinant of survival. Young patients have a good outcome [4] Kim et al in their study on brainstem haemorrhage showed long term survival in these patients [10].

[11] Hashimoto et al., have described 21 brainstem lesions out of a study of 239 traumatic brain injury cases, out of which 5 were 'pure brainstem lesions'. How these cases were managed has not been enumerated (with respect to intensive care management), although an unfavorable outcome of all these brainstem cases, particularly when more than one in number has been mentioned.

[12] In another Tunisian study by Bahloul M et al., 40 cases had normal CT scans & 50 (EDH, n=30; SDH, n=6; lobectomy, n=3; depressed skull #, n=10 & DC, n=1) required surgical intervention, out of 276 child victims of traumatic brain injury. Though all the patients were treated in intensive care set up, data regarding primary brain stem lesions managed is lacking.

## Conclusion

The severe morbidity and mortality associated with BSL mandates management in ICU for round the clock monitoring. This adds to the crunch of bed occupancy in any public sector tertiary care centre, but nevertheless, the outcome of such monitoring is matchless in terms of improved survival & better quality of life (QOL). These cases need further randomized trial study to explore new modalities of treatment protocol in near future. This may be the first of its kind of case report (after extensive search in Pubmed, using different relevant keywords, amongst the English literature) on this prickly issue, while choosing between catering to all & providing best available care to all.

## Acknowledgement

### Conflict of Interest

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

### Patient Consent

The patient /next of kin/guardian has consented to the submission of the case report for submission to the journal.

### Consent to publish

Consent was obtained to use the images included in the manuscript.

## References

1. Tsunoda T, Maeshima S, Watanabe M, Nagai A, Ueno Y, et al. Rehabilitation for a patient with Hemiplegia, Ataxia, Cognitive Dysfunction Caused by Pontine Hemorrhage. *Case Rep Neurol*. 2015 Sep-Dec;7(3):213-220.
2. Samudra N, Figueroa S. Intractable Central Hyperthermia in the setting of Brainstem Hemorrhage. *Ther Hypothermia Temp Manag*. 2016 Jun;6(2):98-101.
3. Tintore M, Rovira A, Mitjana R, Rio J, Auger C, et al. Brainstem lesions in isolated clinically isolated syndromes. *Neurology*. 2010 Nov;75(21):1933-1938.
4. Mahapatra AK, Tandon PN, Banerji AK. Brainstem haemorrhage. A review of 70 ventricular cases. *Abstract NSI meeting 1990, December at Indore*. 1990
5. Tasi FY, Teal JS, Quinn MF, Ahmadi J, Segall HD, et al. CT of brainstem injury. *AJR Am J Roentgenol*. 1980 Apr;134(4):717-723.

6. Texier PH, Diebler C, Bruguier A, Ponsot G Haematoma of brainstem in childhood. *Neuroradiology*. 1984;26(6):499-502.
7. Crompton MR Brainstem lesion after head injury. *Lancet*. 1971;1:669-673.
8. Lindenberg R, Freytag E Brainstem lesion characteristic of traumatic hyperextension of head. *Acta Pathol*. 1970 Dec;90(6):509-515.
9. Gruskiewicz J, Doran Y, Peyser E Recovery from severe craniocerebral injury with brainstem lesion in childhood. *Surg Neurol*. 1973;1:197.
10. Kim RC, Fagin K, Choi BH Prolonged survival after severe traumatic injury limited to brain stem. *Surg Neurol*. 1985 May;23(5):525-528.
11. Hashimoto T, Nakamura N, Richard KE, Frowein RA. Primary brain stem lesions caused by closed head injuries. *Neurosurgical*. 1993;16(4):291-298.
12. Bahloul M, Chelly H, Chaari A, Herguefi L, Dammak H, et al. Isolated traumatic head injury in children: Analysis of 276 observations. *J Emerg Trauma Shock*. 2011 Jan-Mar;4(1):29-36.